

PostScript

LETTERS

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Vitamin K deficient bleeding in cystic fibrosis

We would like to report a female infant (initially breast fed and subsequently formula fed) who had received two 1 mg doses of vitamin K orally, and presented at 9 weeks of age with large haematomas at the sites of her primary immunisations. Her weight had dropped from the 25th to 50th centile at birth to the 2nd centile.

Her haemoglobin was 720 g/l, white blood cell count 13×10^9 /l, platelet count 523×10^9 /l, prothrombin time >10 seconds (normal range 0.8-1.2), activated partial thromboplastin time 109.4 seconds (normal range 24.0-34.0), and fibrinogen 4.5 g/l (normal range 1.7-4.5). She received 1 mg of vitamin K intravenously and repeat coagulation screen was then normal. Sweat osmolality was 110 mmol/l (normal range 17-80) and 105 mmol/l on repeat testing. No chymotrypsin activity was found in the faeces. DNA analysis confirmed homozygosity for delta F 508.

Vitamin K deficiency can occur in undiagnosed cystic fibrosis (CF) infants due to malabsorption of fat soluble vitamins. It is uncommon, since vitamin K is given to all newborns in the UK. As universal screening for CF is not undertaken in the UK, asymptomatic CF patients can be missed and a bleeding diathesis may be the presenting symptom.

Torstenson and colleagues¹ reported three cases of severe life threatening bleeding subsequently diagnosed as CF in infants less than 6 months of age, and Rashid and colleagues² found that 78% of pancreatic insufficient patients had PIVKA-II concentrations >3 µg/l.

Deficiency of vitamin K in children with CF may be due to inadequate dietary intake, maldigestion, and malabsorption.³ Decreased intestinal synthesis of vitamin K₂ following diarrhoeal disease or antibiotic administration can also be a contributing factor.

Our patient developed vitamin K deficient coagulopathy despite receiving oral supplementation and vitamin K from formula feed. The vitamin K deficiency can be attributed to malabsorption secondary to CF and emphasises the need to consider CF as a differential diagnosis in bleeding diathesis

presenting in the first year of life. If a universal neonatal screening programme for diagnosing CF had been in place, a potentially life threatening complication may have been prevented.

T Verghese, D Beverley

York District Hospital, Wigginton Road, York YO31 3HE, UK; David.W.Beverley@york.nhs.uk

References

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- 2 **Rashid M**, Durie P, Andrew M, *et al*. Prevalence of vitamin K deficiency in cystic fibrosis. *Am J Clin Nutr* 1999;**70**:378-82.
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Improving mental health through parenting programmes: are the results valid?

We read the article by Patterson *et al* with interest.¹ Firstly, the percentage of questionnaires returned from the survey should have been 61.8% not 70%, as reported.

Secondly, mental health problems are prevalent in people of lower socioeconomic class. Unfortunately, working class parents were seriously under-represented in the study. The results from educated and predominantly caucasian people from Oxford are not applicable to areas like ours. In the Camden and Islington boroughs of London, we work with parents of mostly lower socioeconomic class and of varied ethnicity—from Albania to Zaire—to whom these results are not relevant. We need more studies conducted in these people to know the best evidence.

Thirdly, the intervention effect is seen at 6 months (short term) follow up. We wonder whether the maturational effect seen in the control group will actually decrease the effect of parenting in the intervention group in the long term? Moreover the intervention effect is said to be statistically significant. But is it clinically significant as well? And there is no cost-benefit analysis given.² Does this justify the considerable use of resources, especially in today's cash strapped, staff depleted (fewer health visitors) NHS? Furthermore, parents in the intervention group might have believed that the parenting programme is efficacious, and consequently feel and perform better than those who were in the control group, as they were aware of group allocation.³ Also, unblinded study personnel who are measuring and recording outcomes (such as quality of life) may provide different interpretation of marginal findings, which can distort the results.³ We now know that negative, inconsistent parental behaviour in families with high levels of adversity are associated with emergence of problems in early childhood and later life.⁴ Hence, we believe that parenting interventions should be applied in high risk populations. That is parents of children with ECBI scores of 127 or more and not children with 100 and above as included in the study.¹ It would have

been helpful if authors gave ECBI and SDQ scales as a web supplement to the above article.

S S Gada, S Kanumakala

Department of Paediatrics, The Royal Free Hospital, Pond Street, London NW3 2QG, UK

Correspondence to Dr Gada; sri@doctors.org.uk

References

- 1 **Patterson J**, Barlow J, Mockford C, *et al*. Improving mental health through parenting programmes: block randomised controlled trial. *Arch Dis Child* 2003;**87**:472-7.
- 2 **Sackett DL**, Straus SE, Richardson WS, *et al*. Evidence-Based Medicine. How to Practice and Teach EBM. London: Churchill Livingstone, 2000.
- 3 **The Evidence-Based Medicine Working Group**. Users' Guide to the Medical Literature: Essentials of Evidence-Based Clinical Practice. Guyatt G, Rennie D, eds. American Medical Association, 2001.
- 4 **Barlow J**. Parenting Programmes, Cochrane protocol. The Cochrane Library, Issue 4. Oxford: Update Software Ltd, 2002.

Authors' reply

Drs Srinivas, Gada, Shanker, and Kanumaka make a number of useful points about our trial. Firstly, they query our response rate. This rate can be calculated using either the number of families or the number of children as the denominator. The rate we quoted 800/1155 is the proportion of families responding. The rate of 61.8% (1105/1788) relates to the proportion of children. Given that this was a trial about parents and parenting we decided that the family based response rate was the most appropriate to report.

Secondly, they point out that this trial was carried out in Oxford and that the socioeconomic mix was somewhat biased towards middle class parents. Although all social groups were well represented in the trial, the point Dr Srinivas and colleagues make is valid. However, behaviour problems are common in all social groups,¹ and because of the distribution of children in each social class, there are considerably more children with behaviour problems in middle class families than there are in families living in social deprivation.² An important finding in this trial was that those who consented to take part were more likely than those who did not to have a child with problem behaviour. We feel that this validates our population approach. At the same time, it is true that our results may not be totally transferable to Islington. That does not stop them, however, being both valid and important.

Dr Srinivas says that more studies of programmes with parents from lower socioeconomic groups are needed. In fact, the great majority of trials of parenting programmes have been conducted with high risk groups and we know from these trials that they are valuable with families living in social deprivation.^{1,4} We are currently completing a systematic review of parenting programmes for minority ethnic families and have found no evidence that parenting programmes are less effective with parents from such groups than they are with those from majority ethnic groups.⁵